Structured relaxation in the treatment of akathisia: case series

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Purpose: Akathisia remains a common side effect especially from antipsychotic medication. If the condition is diagnosed the management options are limited.

Subjects/methodology: We tested a structured relaxation program on nine patients with a diagnosis of schizophrenia suffering from akathisia. All patients were rated on Barnes Akathisia Scale (BAS) before the relaxation program, immediately after and again one week later.

Results: The mean BAS score was before the relaxation 3.3 which reduced to 1.4 immediately after to finally 1.0 a week later. A Wilcoxon signed ranks test revealed a significant reduction in BAS score from baseline to endpoint (P = 0.026; Z = -2.232) and a highly significant reduction from baseline to follow-up (P = 0.008; Z = -2.636).

Discussion: Although the study has a number of limitations the relaxation program appears to be a promising alternative to traditional treatment of akathisia. The patients appreciated the relaxation session but none of them managed to carry it out on their own without professional encouragement. The findings in this case series warrant further investigation with larger numbers of patients.

Keywords: akathisia, relaxation, antipsychotic drugs, schizophrenia

Background

Akathisia (inability to sit still) is a movement disorder characterized by objective movements and restlessness and/or distress, which is common among patients undergoing treatment with psychotropic drugs. It is generally agreed that the syndrome of akathisia comprises both an objective and a subjective component.¹ Although one of the main motivating factors for the development of second generation antipsychotics was to reduce the incidence and severity of treatment-emergent akathisia and other extrapyramidal side effects, these remain problematic and not uncommon:² furthermore, akathisia can also be associated with antidepressant treatment.³

Although often extremely upsetting, the widely held view of an association between akathisia and suicidal behavior has not been proven in larger methodologically sound studies,⁴ and is mainly based on case reports. It is nevertheless a distressing problem that can reduce quality of life and may impede treatment compliance in some patients undergoing pharmacological treatment. In an attempt to make sense of the condition some patients develop a dysfunctional interpretation of the stressful symptoms.⁵

There is no general agreement on how to diagnose akathisia, and this has hampered both research and clinical practice.⁶ If akathisia is recognized, current treatment options include reducing the daily dosage or withdrawal of the implicated medication,
or the addition of other medications. The most widely used pharmacological interventions include anticholinergic drugs, beta-blockers, and benzodiazepines, but the evidence base to support these interventions is limited and treatment may entail risks such as development of hypotension, tolerance, and dependence. In fact the only small, randomized controlled trial on biperiden showed no difference compared to saline water in the treatment of akathisia. Some of the most important differential diagnoses of akathisia are the presence of agitation and anxiety symptoms, and these can be helped significantly with structured relaxation programs. However to our knowledge a relaxation approach has not been examined in patients experiencing akathisia associated with antipsychotic drugs, therefore the aim of this short report is to investigate whether a relaxation approach would reduce akathisia in patients suffering from this syndrome.

Case series
Nine patients with a primary diagnosis of schizophrenia and currently experiencing distressing akathisia (measured on the Barnes Akathisia Scale [BAS]) were invited to participate in a structured relaxation program, lasting 12 minutes and consisting of breathing (four exercises) and tension-relaxation (six exercises). The patients were selected specifically because they suffered from akathisia – all participants were receiving, or had recently received, pharmacological interventions for akathisia such as benzodiazepines, beta blockers, or procyclidine (see Table 1) leading to an unsatisfactorily response. However, the responsible clinicians had been reluctant to lower the dose of antipsychotic medication due to fears of deterioration of psychotic symptoms. Each exercise lasted just over one minute. No patient declined the offer of this intervention. The program was directed by TL and FT. One patient (number 9) underwent a repeat of the relaxation program with TL during the week before the final follow-up assessment.

Results
All patients were rated (by LKH) using the BAS, before the intervention (baseline, BAS median, 3), after the relaxation session (endpoint, BAS median 2), and again one week later (follow-up BAS median, 2). The short follow-up period was decided on to minimize the risk of changes to the medication

Table 1 Demographic and clinical features, and effects of intervention

<table>
<thead>
<tr>
<th>Patient</th>
<th>Group or individual intervention</th>
<th>Gender</th>
<th>Age</th>
<th>Psychotropic or other medication</th>
<th>Baseline BAS score</th>
<th>Endpoint BAS score</th>
<th>Follow-up BAS score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Group</td>
<td>M</td>
<td>46</td>
<td>Flupentixol 9 mg, Amisulpride 400 mg, Procyclidine 10 mg</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>Group</td>
<td>M</td>
<td>22</td>
<td>Olanzapine 20 mg</td>
<td>4</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Group</td>
<td>M</td>
<td>29</td>
<td>Olanzapine 30 mg, Levopromazine 50 mg, Temesta 3 mg</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>Group</td>
<td>M</td>
<td>59</td>
<td>Pipamperone 120 mg, Olanzapine 7.5 mg, Risperdone consta 50 mg, Procyclidine 10 mg, Tranxene 30 mg</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>Group</td>
<td>M</td>
<td>47</td>
<td>Quetiapine 600 mg</td>
<td>3</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>Group</td>
<td>M</td>
<td>29</td>
<td>Prothipendyl 40 mg, Olanzapine 20 mg, Risperdone 4 mg, Procyclidine 15 mg</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>Group</td>
<td>F</td>
<td>31</td>
<td>Risperdone consta 37.5 mg, Aripiprazole 15 mg, Alprazolam 1 mg</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>Group</td>
<td>M</td>
<td>31</td>
<td>Amisulpride 800 mg, Aripiprazole 15 mg</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>9</td>
<td>Individual</td>
<td>F</td>
<td>37</td>
<td>Clozapine 600 mg, Pipamperone 120 mg, Olanzapine 7.5 mg, Levomepromazine 25 mg, Biperidin hydrochloride 2 mg</td>
<td>4</td>
<td>4</td>
<td>1</td>
</tr>
</tbody>
</table>

Abbreviation: BAS, Barnes Akathisia Scale.
regime that could have made the patients unsuitable for the study. All patients were given a written version of the program following the sessions, but according to self-reports, none managed to undertake the program on their own in the week before follow-up. There were no changes to medication regimes in any patient, during the study period.

A Wilcoxon signed rank test showed a significant reduction in BAS score from baseline to endpoint ($P = 0.026, Z = -2.232$) and a highly significant reduction from baseline to follow-up ($P = 0.008; Z = -2.636$). No significant difference was however found between endpoint and follow-up ($P = 0.257; Z = -1.134$). The calculations were carried out on SPSS (version 16.0; SPSS Inc., Chicago, IL).

**Discussion**

These findings are promising, although it is possible that the benefits were at least partly due to factors such as increased attention and greater contact with other patients and staff. Other limitations in this report include the small sample size, preliminary pilot nature of the data, use of a potentially biased (LKH) rater, and the uneven completion of the relaxation program in some patients. Although the number of patients was limited, all but one patient appeared to have benefited from the relaxation program. The approach was received enthusiastically by the participating patients, but no patient managed to perform the program on their own without professional intervention. No detrimental effect was seen in the mental state of any patient.

The structured relaxation program appeared to be well accepted and to be associated with a notable reduction in the severity of symptoms of akathisia in this group of patients with chronic schizophrenia, treated with antipsychotic drugs. The pathophysiology underlying akathisia is not fully understood, and current treatment approaches are less than ideal. It is important to develop evidence-based alternatives to current interventions that are feasible in routine clinical practice. The mechanism underlying the reduction in symptoms of akathisia with the structured relaxation program is uncertain but biofeedback methods may be at least partly responsible for the beneficial effect. Biofeedback is a process that enables a person to change physiological activity (e.g., heart rate, muscle activity). It is conceivable that the relaxation program improved the patients’ ability to minimize the restlessness characteristic of akathisia. The sustained effect at 1-week follow-up is intriguing and warrants further study, possibly comparing structured relaxation with current pharmacological treatment options, such as use of beta-blockers or benzodiazepines in an open label clinical trial.

**Disclosures**

LKH has received speaker-fees from most of the major pharmaceutical companies such as Bristol-Meyers-Squibb, Janssen-Cilag, Astra-Zeneca, Wyeth and Lily. TL and FT have no disclosures to make. DSB has acted as a consultant to a number of pharmaceutical companies (Asahi, AstraZeneca, Cephalon, Eli Lilly, GSK, Lundbeck, Organon, Pharmacia, Pierre Fabre, Pfizer, Roche, Servier, Sumitomo, Wyeth) and holds or has held research grants (on behalf of his employer) from a number of companies (Cephalon, Eli Lilly, GSK, Lundbeck, Organon, Pfizer, Pharmacia, Roche, Wyeth).

**References**